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DATE: Thursday, August 03, 2006

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☐	L1	zmax1 or zmax 1	26

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=> s zmax 1 or zmax1
L1          53 ZMAX 1 OR ZMAX1

=> s l1 and bone
L2           8 L1 AND BONE

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3           7 DUP REM L2 (1 DUPLICATE REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 7 ANSWERS -
CONTINUE? Y/(N):y

L3  ANSWER 1 OF 7  BIOSIS  COPYRIGHT (c) 2006 The
Thomson Corporation  on STN
    DUPLICATE 1
AN  2004:383095  BIOSIS <<LOGINID::20060803>>
DN  PREV200400388096
TI  High  ***bone***  mass gene of 1.1q13.3.
AU  Carulli, John P. [Inventor, Reprint Author]; Little, Randall D.
    [Inventor]; Recker, Robert R. [Inventor]; Johnson, Mark L.
[Inventor]
CS  ASSIGNEE: Genome Therapeutics Corporation
PI  US 6780609 20040824
SO  Official Gazette of the United States Patent and Trademark
Office Patents,
    (Aug 24 2004) Vol. 1285, No. 4.
http://www.uspto.gov/web/menu/patdata.html
    . e-file.
    ISSN: 0098-1133 (ISSN print).
DT  Patent
LA  English
ED  Entered STN: 29 Sep 2004
    Last Updated on STN: 29 Sep 2004
AB  The present invention relates to methods and materials used to
isolate and
    detect a high  ***bone***  mass gene and a corresponding wild-
type gene,
    and mutants thereof.  The present invention also relates to the
high
     ***bone***  mass gene, the corresponding wild-type gene, and
mutants
    thereof.  The genes identified in the present invention are
implicated in
     ***bone***  development.  The invention also provides nucleic
acids,
    including coding sequences, oligonucleotide primers and probes,
proteins,
    cloning vectors, expression vectors, transformed hosts, methods
of
    developing pharmaceutical compositions, methods of identifying
molecules
    involved in  ***bone***  development, and methods of
diagnosing and
    treating diseases involved in  ***bone***  development.  In
preferred
    embodiments, the present invention is directed to methods for
treating,
    diagnosing and preventing osteoporosis.

L3  ANSWER 2 OF 7  BIOSIS  COPYRIGHT (c) 2006 The
Thomson Corporation  on STN
AN  2004:351355  BIOSIS <<LOGINID::20060803>>
DN  PREV200400354810
TI  High  ***bone***  mass gene of 11q13.3.
AU  Carulli, John P. [Inventor, Reprint Author]; Little, Randall D.
    [Inventor]; Recker, Robert R. [Inventor]; Johnson, Mark L.
[Inventor]
CS  Southboro, MA, USA
    ASSIGNEE: Genome Therapeutics Corporation; Creighton
University School of
    Medicine, Omaha, NE, USA
PI  US 6770461 20040803
SO  Official Gazette of the United States Patent and Trademark
Office Patents,
    (Aug 3 2004) Vol. 1285, No. 1.
http://www.uspto.gov/web/menu/patdata.html.
    e-file.
    ISSN: 0098-1133 (ISSN print).
DT  Patent
LA  English
ED  Entered STN: 26 Aug 2004
    Last Updated on STN: 26 Aug 2004
AB  The present invention relates to methods and materials used to
isolate and
    detect a high  ***bone***  mass gene and a corresponding wild-
type gene,
    and mutants thereof.  The present invention also relates to the
high
     ***bone***  mass gene, the corresponding wild-type gene, and
mutants
    thereof The genes identified in the present invention are
implicated in
```

bone development. The invention also provides nucleic acids,
including coding sequences, oligonucleotide primers and probes, proteins,
cloning vectors, expression vectors, transformed hosts, methods of
developing pharmaceutical compositions, methods of identifying molecules
involved in ***bone*** development, and methods of diagnosing and
treating diseases involved in ***bone*** development. In preferred
embodiments, the present invention is directed to methods for treating,
diagnosing and preventing osteoporosis.

L3 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2002:888494 CAPLUS <<LOGINID::20060803>>
DN 137:381503
TI Compositions and methods for modulating Dkk-mediated protein interactions
and their diagnostic and therapeutic uses
IN Allen, Kristina; Anisowicz, Anthony; Bhat, Bheem M.; Damagnez, Veronique;
Robinson, John Allen; Yaworsky, Paul J.
PA Genome Therapeutics Corporation, USA; Wyeth, John and Brother Ltd.
SO PCT Int. Appl., 376 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002092015	A2	20021121	WO 2002-US15982	20020517
WO 2002092015	A3	20031023		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2446582	AA	20021121	CA 2002-2446582	20020517
EP 1395285	A2	20040310	EP 2002-744162	20020517
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2002009836	A	20041207	BR 2002-9836	20020517
JP 2005512508	T2	20050512	JP 2002-588934	20020517
US 2004038860	A1	20040226	US 2002-182936	20020802
PRAI US 2001-291311P	P	20010517		
US 2002-353058P	P	20020201		
US 2002-361293P	P	20020304		
WO 2002-US15982	W	20020517		

AB The present invention provides reagents, compds., compns., and methods
relating to interactions of the extracellular domain of LRP5/***ZMax1***
, HBM (a variant of LRP5), and/or LRP6 with Dkk, including Dkk-1. The
various nucleic acids, polypeptides, antibodies, assay methods, diagnostic
methods, and methods of treatment of the present invention are related to
and impact on Dkk, LRP5, LRP6, HBM, and Wnt signaling. The invention
claims sequences for peptide aptamers which bind to LRP5 or Dkk-1 and
sequences for Dkk-1 peptides which are recognized by antibodies. HBM is a
Gly171Val polymorphism in LDL receptor-related protein LRP5/Zmax, which
has been identified as conferring a high ***bone*** mass phenotype in
a population of related humans. The protein dickkopf-1 (Dkk-1) is
required for head formation in early development and murine limb
morphogenesis and is reported to be an antagonist of the Wnt signaling
pathway. Dkk-1 protein interacts with the ligand-binding domain of LRP5.
Dkk-1 also binds to LRP6, but the EGF repeat domains of LRP6 rather than
the ligand-binding domain are required for interaction. Dkk-1 is able to
repress LRP5-mediated Wnt signaling but not HBM-mediated Wnt signaling and
Dkk-1 also inhibits LRP6 activity. LRP5, LRP6, HBM, Dkk and Wnt are
implicated in ***bone*** and lipid cellular signaling. Thus, the
present invention provides reagents and methods for modulating lipid
levels and/or ***bone*** mass and is useful in the treatment and
diagnosis of abnormal lipid levels and ***bone*** mass disorders, such
as osteoporosis. Examples of the invention include a yeast two-hybrid
screen for Dkk-1 interacting proteins, generation of LRP5
polymorphism-specific antibodies and Dkk-1 specific antibodies, effects of
exogenous Dkk-1 on Wnt-mediated signaling in the Xenopus embryo assay, and
effects of recombinant Dkk and Wnt3a/1 on TCF-luciferase reporter gene
expression in human cell lines with endogenous LRP5/6.

L3 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2002:888480 CAPLUS <<LOGINID::20060803>>
DN 137:380994
TI High ***bone*** mass variants of the human ***Zmax1*** /LRP5 gene
modulate ***bone*** mass and lipid levels
IN Allen, Kristina; Anisowicz, Anthony; Graham, James R.; Morales, Arturo;
Yaworsky, Paul J.; Liu, Wei
PA Genome Therapeutics Corporation, USA; Wyeth, John and Brother Ltd.
SO PCT Int. Appl., 629 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002092000	A2	20021121	WO 2002-US14877	20020513
WO 2002092000	A3	20041007		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2446821	AA	20021121	CA 2002-2446821	20020513
BR 2002009563	A	20041207	BR 2002-9563	20020513
EP 1483288	A2	20041208	EP 2002-746370	20020513
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004537289	T2	20041216	JP 2002-588919	20020513
US 2005070699	A1	20050331	US 2004-477173	20041104
PRAI US 2001-290071P	P	20010511		
US 2001-291311P	P	20010517		
US 2002-353058P	P	20020201		
US 2002-361293P	P	20020304		
WO 2002-US14877	W	20020513		

AB The present invention relates to methods and materials used to express an

HBM-like polypeptide derived from HBM (high ***bone*** mass), LRP5 or
LRP6 in animal cells and transgenic animals. The HBM gene comprises 23
exons on human chromosome 11q13.3, and is shown to be an allele of the
Zmax1 /LRP5 gene; a variety addnl. single nucleotide polymorphisms
are also identified. The ***Zmax1*** (LRP5) protein with a
glycine-170-valine substitution causes a HMB phenotype involving high
bone mass and modified lipid levels, whereas the valine-170
isoform does not. This mutation is in the propeller 1 domain of the
protein, and modulates Wnt signaling, Dkk activity, and/or LRP5/6
activity. The present invention also relates to transgenic animals
expressing the HBM-like polypeptides. The invention provides nucleic
acids, including coding sequences, oligonucleotide primers and probes,
proteins, cloning vectors, expression vectors, transformed hosts, methods
of developing pharmaceutical compns., methods of identifying mols.
involved in ***bone*** development, and methods of diagnosing and
treating diseases involved in ***bone*** development and lipid
modulation. In preferred embodiments, the present invention is directed
to methods for treating, diagnosing and preventing osteoporosis.

L3 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:886643 CAPLUS <<LOGINID::20060803>>
DN 136:32816
TI Regulating lipid levels via the human ***Zmax1*** or high-***bone***
-mass HBM gene
IN Carulli, John P.; Little, Randall D.; Recker, Robert R.; Johnson, Mark L.
PA Genome Therapeutics Corporation, USA; Creighton University School of
Medicine
SO PCT Int. Appl., 409 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001092891	A2	20011206	WO 2001-US16946	20010525
WO 2001092891	A3	20020725		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2410253	AA	20011206	CA 2001-2410253	20010525
AU 2001269712	A2	20011211	AU 2001-269712	20010525
EP 1285002	A2	20030226	EP 2001-948240	20010525
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2001011057	A	20030415	BR 2001-11057	20010525
JP 2004523724	T2	20040805	JP 2002-501047	20010525
NZ 522600	A	20040924	NZ 2001-522600	20010525
ZA 2002008977	A	20031105	ZA 2002-8977	20021105

PRAI US 2000-578900 A 20000526
WO 2001-US16946 W 20010525
AB The present invention relates to the high ***bone*** mass (HBM) gene,
the corresponding wild-type gene (***Zmax1***), and mutants thereof.
The ***Zmax1*** /HBM gene was located on chromosome 11q13.3 by genetic
linkage and mutation anal. Cloning methods using bacterial artificial
chromosomes enabled focus on the chromosome region of 11q13.3 and
sequencing of the autosomal dominant gene. A guanine-to-thymine
polymorphism at position 582 (glycine-to-valine at position 171 in the
protein)in ***Zmax1*** gene produces the HBM gene and the HBM
phenotype as well as altered lipid levels. Hybridization for
Zmax1 is primarily detected in areas of ***bone*** involved in
remodeling, including the endosteum and trabecular ***bone*** within
the metaphysis; pos. signals are also obsd in selected ***bone***
lining cells of the periosteum and epiphysis and in chondrocytes within
the growth plate. The genes identified in the present invention are
implicated in regulation of physiol. lipid levels, and thereby
lipid-mediated diseases and conditions. The invention also provides
nucleic acids, including coding sequences, oligonucleotide primers and
probes, proteins, cloning vectors, expression vectors, transformed hosts,
methods of developing pharmaceutical compns., methods of identifying mols.
involved in lipid level regulation in a subject. In preferred
embodiments, the present invention is directed to methods for treating and
preventing atherosclerosis, arteriosclerosis cardiovascular disease,
atherosclerotic and arteriosclerotic assocd. conditions.

L3 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:763189 CAPLUS <<LOGINID::20060803>>
DN 135:328141
TI Human gene ***Zmax1*** of 11q13.3, HBM (high ***bone*** mass)
allele, encoded polypeptides, and their diagnostic and therapeutic uses
IN Carulli, John P.; Little, Randall D.; Recker, Robert R.; Johnson, Mark L.
PA Genome Therapeutics Corporation, USA
SO PCT Int. Appl., 443 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001077327	A1	20011018	WO 2000-US16951	20000621
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6770461	B1	20040803	US 2000-544398	20000405
US 6780609	B1	20040824	US 2000-543771	20000405
CA 2402410	AA	20011018	CA 2000-2402410	20000621
EP 1268775	A1	20030102	EP 2000-941578	20000621
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2004515209	T2	20040527	JP 2001-575181	20000621
NZ 521769	A	20041224	NZ 2000-521769	20000621

US 2003219793 A1 20031127 US 2003-374979 20030228
PRAI US 2000-543771 A 20000405
US 2000-544398 A 20000405
US 1998-71449P P 19980113
US 1998-105511P P 19981023
US 1999-229319 A2 19990113
US 2000-578900 W 20000526
WO 2000-US16951 W 20000621
US 2002-240851 A1 20021004
AB The present invention relates to methods and materials used to isolate and
detect a high ***bone*** mass gene and a corresponding wild-type gene,
and mutants thereof. The present invention also relates to the high
bone mass allele, the corresponding wild-type gene, ***Zmax1***
, and mutants thereof. The genes identified in the present invention are
implicated in ***bone*** development and in focal adhesion signaling.
The invention also provides nucleic acids, including coding sequences,
oligonucleotide primers and probes, proteins, cloning vectors, expression
vectors, transformed hosts, methods of developing pharmaceutical comps.,
methods of identifying mols. involved in ***bone*** development, and
methods of diagnosing and treating diseases involved in ***bone***
development. In preferred embodiments, the present invention is directed
to methods for treating, diagnosing and preventing osteoporosis. The
invention describes expanded pedigree anal. and genetic linkage anal. of a
high ***bone*** mass (HBM) gene now known as an allele of human gene
Zmax1 . Older individuals with the HBM allele do not show loss of
bone mass compared to normal individuals, do not have
osteoporosis, and do not have any known high ***bone*** mass syndrome.
Gene ***Zmax1*** was localized between genetic markers on human
chromosome 11q13.3 and subsequently, BAC clones with the gene were
sequenced. The HBM allele is inherited as an autosomal dominant gene and
is a G .fwdarw. T mutation at nucleotide 582 in exon 3 which results in a
G171V substitution in the encoded protein. Addnl. genotyping of 911
individuals established that the HBM allele is rare and never found in
unaffected individuals. "Silent" SNPs (single nucleotide polymorphisms)
in the gene ***Zmax1*** region were also identified. Gene
Zmax1 encodes an LDL-receptor-related protein and the HBM mutation
occurs in a conserved region of the presumed extracellular domain.
Proteins interacting with the cytoplasmic domain of gene ***Zmax1***
protein in a yeast two-hybrid assay were identified and include many
proteins found at cell-cell and cell-matrix contact sites. These results
suggest a potential role for gene ***Zmax1*** in focal adhesion
signaling and suggest that regulating gene ***Zmax1*** expression or
protein binding may affect ***bone*** processes.
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 1986:319794 BIOSIS <<LOGINID::20060803>>
DN PREV198682044099; BA82:44099
TI ASSIGNMENT OF THE GENE FOR DYSKERATOSIS CONGENITA TO XQ28.
AU CONNOR J M [Reprint author]; GATHERER D; GRAY F C; PIRRIT L A; AFFARA N A
CS UNIVERSITY DEPARTMENT OF GENETICS, DUNCAN GUTHRIE INSTITUTE OF MEDICAL
GENETICS, YORKHILL, GLASGOW, G3 9SJ, UK
SO Human Genetics, (1986) Vol. 72, No. 4, pp. 348-351.
CODEN: HUGEDQ. ISSN: 0340-6717.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 8 Aug 1986
Last Updated on STN: 8 Aug 1986
AB Dyskeratosis congenita is an X-linked recessive disorder with diagnostic
dermatological features, ***bone*** marrow hypofunction, and a
predisposition to neoplasia in early adult life. Linkage analysis was
undertaken in an extensive family with the condition using the Xg blood
group and 17 cloned X chromosomal DNA sequences which recognise
restriction fragment length polymorphisms (RFLPs). No recombination was
observed between the locus for dyskeratosis congenita (DKC) and the RFLPs
identified by DXS52 (St 14-1) (Zmax = 3.33 at .THETA.max = 0 with 95%
confidence limits of 0 to 14 cM). Similarly no recombination was observed
for the disease locus and F8 (***Zmax*** = ***1*** .23 at
.THETA.max = 0) nor for DXS15 (***Zmax*** = ***1*** .62 at
.THETA.max = 0), but both of these markers were only informative in part
of the family whereas DXS52 was fully informative. DXS52, DXS15, and F8
are known to be tightly linked and have previously been assigned to Xq28.
Thus the gene for dyskeratosis congenita can be assigned to Xq28. These
DNA sequence polymorphisms will be of clinical value for carrier detection
and prenatal diagnosis.

=> s carulli, j?/au
L4 85 CARULLI, J?/AU

=> s Little, r?/au
L5 2133 LITTLE, R?/AU

=> s recker, r?/au
L6 674 RECKER, R?/AU

=> s johnson, m?/au
L7 20657 JOHNSON, M?/AU

=> s l4 or l5 or l7 or l6
L8 23482 L4 OR L5 OR L7 OR L6

=> s l8 and (zmax or hbm)
L9 20 L8 AND (ZMAX OR HBM)

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10 11 DUP REM L9 (9 DUPLICATES REMOVED)

=> d bib abs 1-
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L10 ANSWER 1 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
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AN 2006250138 EMBASE <<LOGINID::20060803>>
TI LRP5: Structural and molecular aspects.
AU ***Johnson M.L.***
CS Dr. M.L. Johnson, Department of Oral Biology, UMKC School of Dentistry,
650 East 25th Street, Kansas City, MO 64108, United States.
johnsonmark@umkc.edu
SO Clinical Reviews in Bone and Mineral Metabolism, (2006) Vol. 4, No. 2, pp.
97-106. .
Refs: 67
ISSN: 1534-8644 CODEN: CRBMBF
CY United States
DT Journal; General Review
FS 022 Human Genetics
030 Pharmacology
033 Orthopedic Surgery
037 Drug Literature Index
LA English
SL English
ED Entered STN: 13 Jun 2006
Last Updated on STN: 13 Jun 2006
AB Several lines of evidence have provided compelling support for low-density
lipoprotein receptor-related protein 5 (LRP5) and the canonical
Wnt/.beta.-catenin signaling pathway as being important and essential for

bone formation. Lrp5 and its close homolog, Lrp6, are coreceptors with
frizzled for Wnt proteins. Binding of Wnt proteins to Lrp5/6 and frizzled
activates the Wnt/.beta.-catenin signaling pathway. Mutations in Lrp5
have been shown to give rise to human diseases of low bone mass and loss
of vision such as osteoporosis pseudoglioma syndrome (OPPG) and familial
exudative vitreoretinopathy (FEVR) as well as several human conditions
with increased bone mass and reduced fracture risk, such as the high bone
mass (***HBM***) phenotype. Although it is well established that the
Lrp5/6-Wnt canonical pathway is important in embryonic growth and
development of the skeleton, its role in the adult skeleton is not clear.
Accumulating evidence now supports an important role for Lrp5 in the
response of the postnatal skeleton to mechanical load. Transgenic mice
carrying the human ***HBM*** mutation (LRP5(G171V)) have increased
sensitivity to load, and mice lacking Lrp5 do not respond to mechanical
load. In vivo loading of LRP5(G171V) mice tibia results in increased
osteoprotegerin (OPG) gene expression. Mice with either gain- or
loss-of-function mutations in protein components of the canonical pathway
below the level of Lrp5/6 develop high or low bone mass mainly as a
consequence of altered OPG production by osteoblasts, which subsequently
alters osteoclastogenesis. Thus, activation of the canonical Wnt
signaling pathway apparently has multiple modes of action on bone cells to
regulate bone mass. Given the clear importance of LRP5 in regulating bone
mass, this gene/protein represents a potentially exciting new target for
the development of anabolic agents to treat osteoporosis.

L10 ANSWER 2 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 2
AN 2004:383095 BIOSIS <<LOGINID::20060803>>
DN PREV200400388096
TI High bone mass gene of 1.1q13.3.
AU ***Carulli, John P.*** [Inventor, Reprint Author]; ***Little,*** *** Randall D.*** [Inventor]; ***Recker, Robert R.*** [Inventor];
Johnson, Mark L. [Inventor]
CS ASSIGNEE: Genome Therapeutics Corporation
PI US 6780609 20040824
SO Official Gazette of the United States Patent and Trademark Office Patents,
(Aug 24 2004) Vol. 1285, No. 4.
http://www.uspto.gov/web/menu/patdata.html
. e-file.
ISSN: 0098-1133 (ISSN print).
DT Patent
LA English
ED Entered STN: 29 Sep 2004
Last Updated on STN: 29 Sep 2004
AB The present invention relates to methods and materials used to isolate and
detect a high bone mass gene and a corresponding wild-type gene, and
mutants thereof. The present invention also relates to the high bone mass
gene, the corresponding wild-type gene, and mutants thereof. The genes
identified in the present invention are implicated in bone development.
The invention also provides nucleic acids, including coding sequences,
oligonucleotide primers and probes, proteins, cloning vectors, expression
vectors, transformed hosts, methods of developing pharmaceutical
compositions, methods of identifying molecules involved in bone
development, and methods of diagnosing and treating diseases involved in
bone development. In preferred embodiments, the present invention is
directed to methods for treating, diagnosing and preventing osteoporosis.

L10 ANSWER 3 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 3
AN 2004:351355 BIOSIS <<LOGINID::20060803>>
DN PREV200400354810
TI High bone mass gene of 11q13.3.
AU ***Carulli, John P.*** [Inventor, Reprint Author]; ***Little,*** *** Randall D.*** [Inventor]; ***Recker, Robert R.*** [Inventor];
Johnson, Mark L. [Inventor]
CS Southboro, MA, USA
ASSIGNEE: Genome Therapeutics Corporation; Creighton University School of
Medicine, Omaha, NE, USA
PI US 6770461 20040803
SO Official Gazette of the United States Patent and Trademark Office Patents,
(Aug 3 2004) Vol. 1285, No. 1.
http://www.uspto.gov/web/menu/patdata.html.
e-file.
ISSN: 0098-1133 (ISSN print).
DT Patent
LA English
ED Entered STN: 26 Aug 2004
Last Updated on STN: 26 Aug 2004
AB The present invention relates to methods and materials used to isolate and
detect a high bone mass gene and a corresponding wild-type gene, and
mutants thereof. The present invention also relates to the high bone mass
gene, the corresponding wild-type gene, and mutants thereof The genes
identified in the present invention are implicated in bone development.
The invention also provides nucleic acids, including coding sequences,
oligonucleotide primers and probes, proteins, cloning vectors, expression
vectors, transformed hosts, methods of developing pharmaceutical
compositions, methods of identifying molecules involved in bone
development, and methods of diagnosing and treating diseases involved in
bone development. In preferred embodiments, the present invention is
directed to methods for treating, diagnosing and preventing osteoporosis.

L10 ANSWER 4 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 2005:264978 BIOSIS <<LOGINID::20060803>>
DN PREV200510058184
TI Ulna loading response altered by the ***HBM*** mutation.
AU Cullen, D. M. [Reprint Author]; Akhter, M. P.; ***Johnson, M. L.*** ;
Morgan, S.; ***Recker, R. R.***
CS Creighton Univ, Osteoporosis Res Ctr, Omaha, NE USA
SO Journal of Bone and Mineral Research, (OCT 2004) Vol. 19, pp. S396.
Meeting Info.: 26th Annual Meeting of the American-Society-for-Bone-and-
Mineral-Research. Seattle, WA, USA. October 01 -05, 2004. Amer Soc Bone &
Mineral Res.
CODEN: JBMREJ. ISSN: 0884-0431.
DT Conference; (Meeting)
Conference; (Meeting Poster)
LA English
ED Entered STN: 21 Jul 2005
Last Updated on STN: 21 Jul 2005

L10 ANSWER 5 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
reserved on STN DUPLICATE 4
AN 2004263068 EMBASE <<LOGINID::20060803>>
TI Bone biomechanical properties in LRP5 mutant mice.
AU Akhter M.P.; Wells D.J.; Short S.J.; Cullen D.M.; ***Johnson M.L.*** ;
Haynatzki G.R.; Babij P.; Allen K.M.; Yaworsky P.J.; Bex F.; ***Recker***
*** R.R.***
CS M.P. Akhter, Osteoporosis Research Center, Creighton University, 601
North, 30th Street #4820, Omaha, NE 68131, United States.
akhtermp@creighton.edu
SO Bone, (2004) Vol. 35, No. 1, pp. 162-169. .
Refs: 33

ISSN: 8756-3282 CODEN: BONEDL
PUI S 8756-3282(04)00084-5
CY United States
DT Journal; Article
FS 029 Clinical Biochemistry
033 Orthopedic Surgery
LA English
SL English
ED Entered STN: 9 Jul 2004
Last Updated on STN: 9 Jul 2004
AB The mutation responsible for the high bone mass (***HBM***) phenotype
has been postulated to act through the adaptive response of bone to
mechanical load resulting in denser and stronger skeletons in humans and
animals. The bone phenotype of members of a ***HBM*** family is
characterized by normally shaped bones that are exceptionally dense,
particularly at load bearing sites [Cancer Res. 59 (1999) 1572]. The high
bone mass (***HBM***) mutation was identified as a glycine to valine
substitution at amino acid residue 171 in the gene coding for low-density
lipoprotein receptor-related protein 5 (LRP5) [Bone Miner. Res. 16(4)
(2001) 758]. Thus, efforts have focused on the examination of the role of
LRP5 and the G171V mutation in bone mechanotransduction responses [J.
Bone Miner. Res 18 (2002) 960]. Transgenic mice expressing the human
G171V mutation have been shown to have skeletal phenotypes remarkably
similar to those seen in affected individuals. In this study, we have
identified differences in biomechanical (structural and apparent material)
properties, bone mass/ash, and bone stiffness of cortical and cancellous
bone driven by the G171V mutation in LRP5. As in humans, the LRP5 G171V
plays an important role in regulating bone structural phenotypes in mice.
These bone phenotypes include greater structural and apparent material
properties in ***HBM*** HET as compared to non-transgenic littermates
(NTG) mice. Body size and weight in ***HBM*** HET were similar to
that in NTG control mice. However, the LRP5 G171V mutation in HET mice
results in a skeleton that has greater structural (femoral shaft, femoral
neck, tibiae, vertebral body) and apparent material (vertebral body)
strength, percent bone ash weight (ulnae), and tibial stiffness. Despite
similar body weight to NTG mice, the denser and stiffer bones in G171V
mice may represent greater bone formation sensitivity to normal mechanical
stimuli resulting in an overadaptation of skeleton to weight-related
forces. .COPYRGT. 2004 Elsevier Inc. All rights reserved.

L10 ANSWER 6 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
reserved on STN
AN 2002346039 EMBASE <<LOGINID::20060803>>
TI The gene for high bone mass.
AU ***Johnson M.L.*** ; Picconi J.L.; ***Recker R.R.***
CS Dr. M.L. Johnson, Osteoporosis Research Center, Creighton Univ. School of
Medicine, 601 North 30th Street, Omaha, NE 68131, United States.
MARKL@creighton.edu
SO Endocrinologist, (2002) Vol. 12, No. 5, pp. 445-453. .
Refs: 79
ISSN: 1051-2144 CODEN: EDOCEB
CY United States
DT Journal; General Review
FS 022 Human Genetics
033 Orthopedic Surgery
LA English
SL English
ED Entered STN: 17 Oct 2002
Last Updated on STN: 17 Oct 2002
AB The mass, density, and architecture of the skeleton are adapted to enable
it to perform its mechanical, protective, and metabolic functions.
Osteoporosis is a condition of lost adaptation characterized by decreased
skeletal mass and density and increased skeletal fragility. Many diseases
result in increased bone density, including osteopetrosis and Paget's
disease, but deformities or bony lesions with decreased skeletal integrity
usually accompany these conditions. We have identified a kindred with
high bone mass (***HBM***) yet normally shaped bones. Linkage
analysis localized the gene for the ***HBM*** trait to chromosome 11
(11q12-13). Subsequent physical mapping and mutation analysis have
identified the cause as a point mutation in the LDL receptor-related
protein 5 (Lrp5) gene that results in a valine substitution for glycine at
position 171 in the protein. This protein is important in the Wnt
signaling pathway. The authors have hypothesized that the Lrp5
gene/pathway is part of the mechanism by which bone senses mechanical
load. Increased bone strength, ***HBM*** , and a phenotype resembling
our human kindred develop in transgenic mice carrying the human Lrp5 gene
with the ***HBM*** mutation. Recent data indicate that the
HBM mutation reduces the threshold for response of the skeleton to
mechanical load resulting in an overadaptation to normal mechanical loads.
This discovery has opened the door to understanding one of the most
important paradigms in bone biology, how bones respond and adapt to
mechanical loading. Understanding the mechanosensation pathway and its
regulation will lead us to new treatments for osteoporosis.

L10 ANSWER 7 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 2003:431910 BIOSIS <<LOGINID::20060803>>
DN PREV200300431910
TI Bone sensitivity to mechanical loads with the Lrp5 ***HBM*** mutation.
AU Cullen, D. M. [Reprint Author]; Akhter, M. P. [Reprint Author]; Mace, D.
[Reprint Author]; ***Johnson, M. L.*** [Reprint Author]; Babij, P.;
Recker, R. R. [Reprint Author]
CS Osteoporosis Research Center, Creighton University, Omaha, NE, USA
SO Journal of Bone and Mineral Research, (September 2002) Vol. 17, No. Suppl
1, pp. S332. print.
Meeting Info.: Twenty-Fourth Annual Meeting of the American Society for
Bone and Mineral Research. San Antonio, Texas, USA. September 20-24, 2002.
American Society for Bone and Mineral Research.
ISSN: 0884-0431 (ISSN print).
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 17 Sep 2003
Last Updated on STN: 17 Sep 2003

L10 ANSWER 8 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
reserved on STN DUPLICATE 5
AN 2002013553 EMBASE <<LOGINID::20060803>>
TI A mutation in the LDL receptor-related protein 5 gene results in the
autosomal dominant high-bone-mass trait.
AU ***Little R.D.*** ; ***Carulli J.P.*** ; Del Mastro R.G.; Dupuis J.;
Osborne M.; Folz C.; Manning S.P.; Swain P.M.; Zhao S.C.; Eustace B.;
Lappe M.M.; Spitzer L.; Zweier S.; Braunschweiger K.; Benchekroun Y.; Hu
X.; Adair R.; Chee L.; Fitzgerald M.G.; Tulig C.; Caruso A.; Tzellas N.;
Bawa A.; Franklin B.; McGuire S.; Nogues X.; Gong G.; Allen K.M.;
Anisowicz A.; Morales A.J.; Lomedico P.T.; Recker S.M.; Van Eerdewegh P.;
Recker R.R. ; ***Johnson M.L.***
CS Dr. R.D. Little, Human Genetics Department, Genome Therapeutics

Corporation, 100 Beaver Street, Waltham, MA 02453, United States.
rlittle@genomecorp.com
SO American Journal of Human Genetics, (2002) Vol. 70, No. 1, pp. 11-19. .
Refs: 32
ISSN: 0002-9297 CODEN: AJHGAG
CY United States
DT Journal; Article
FS 022 Human Genetics
LA English
SL English
ED Entered STN: 17 Jan 2002
Last Updated on STN: 17 Jan 2002
AB Osteoporosis is a complex disease that affects .ltoreq.10 million people
in the United States and results in 1.5 million fractures annually. In
addition, the high prevalence of osteopenia (low bone mass) in the general
population places a large number of people at risk for developing the
disease. In an effort to identify genetic factors influencing bone
density, we characterized a family that includes individuals who possess
exceptionally dense bones but are otherwise phenotypically normal. This
high-bone-mass trait (***HBM***) was originally localized by linkage
analysis to chromosome 11q12-13. We refined the interval by extending the
pedigree and genotyping additional markers. A systematic search for
mutations that segregated with the ***HBM*** phenotype uncovered an
amino acid change, in a predicted .beta.-propeller module of the
low-density lipoprotein receptor-related protein 5 (LRP5), that results in
the ***HBM*** phenotype. During analysis of .ltoreq.1,000
individuals, this mutation was observed only in affected individuals from
the ***HBM*** kindred. By use of in situ hybridization to rat tibia,
expression of LRP5 was detected in areas of bone involved in remodeling.
Our findings suggest that the ***HBM*** mutation confers a unique
osteogenic activity in bone remodeling, and this understanding may
facilitate the development of novel therapies for the treatment of
osteoporosis.

L10 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:886643 CAPLUS <<LOGINID::20060803>>
DN 136:32816
TI Regulating lipid levels via the human Zmax1 or high-bone-mass ***HBM***
gene
IN ***Carulli, John P.*** ; ***Little, Randall D.*** ; ***Recker,*** *** Robert R.*** ; ***Johnson, Mark L.***
PA Genome Therapeutics Corporation, USA; Creighton University School of
Medicine
SO PCT Int. Appl., 409 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001092891	A2	20011206	WO 2001-US16946	20010525
WO 2001092891	A3	20020725		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2410253	AA	20011206	CA 2001-2410253	20010525
AU 2001269712	A2	20011211	AU 2001-269712	20010525
EP 1285002	A2	20030226	EP 2001-948240	20010525
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
BR 2001011057	A	20030415	BR 2001-11057	20010525
JP 2004523724	T2	20040805	JP 2002-501047	20010525
NZ 522600	A	20040924	NZ 2001-522600	20010525
ZA 2002008977	A	20031105	ZA 2002-8977	20021105

PRAI US 2000-578900 A 20000526
WO 2001-US16946 W 20010525
AB The present invention relates to the high bone mass (***HBM***) gene,
the corresponding wild-type gene (Zmax1), and mutants thereof. The Zmax1/
HBM gene was located on chromosome 11q13.3 by genetic linkage and
mutation anal. Cloning methods using bacterial artificial chromosomes
enabled focus on the chromosome region of 11q13.3 and sequencing of the
autosomal dominant gene. A guanine-to-thymine polymorphism at position
582 (glycine-to-valine at position 171 in the protein)in Zmax1 gene
produces the ***HBM*** gene and the ***HBM*** phenotype as well as
altered lipid levels. Hybridization for Zmax1 is primarily detected in
areas of bone involved in remodeling, including the endosteum and
trabecular bone within the metaphysis; pos. signals are also obsd in
selected bone lining cells of the periosteum and epiphysis and in
chondrocytes within the growth plate. The genes identified in the present
invention are implicated in regulation of physiol. lipid levels, and
thereby lipid-mediated diseases and conditions. The invention also
provides nucleic acids, including coding sequences, oligonucleotide
primers and probes, proteins, cloning vectors, expression vectors,
transformed hosts, methods of developing pharmaceutical compns., methods
of identifying mols. involved in lipid level regulation in a subject. In
preferred embodiments, the present invention is directed to methods for
treating and preventing atherosclerosis, arteriosclerosis cardiovascular
disease, atherosclerotic and arteriosclerotic assocd. conditions.

L10 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2001:763189 CAPLUS <<LOGINID::20060803>>
DN 135:328141
TI Human gene Zmax1 of 11q13.3, ***HBM*** (high bone mass) allele,
encoded polypeptides, and their diagnostic and therapeutic uses
IN ***Carulli, John P.*** ; ***Little, Randall D.*** ; ***Recker,*** *** Robert R.*** ; ***Johnson, Mark L.***
PA Genome Therapeutics Corporation, USA
SO PCT Int. Appl., 443 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001077327	A1	20011018	WO 2000-US16951	20000621
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,				

DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,
CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
US 6770461 B1 20040803 US 2000-544398 20000405
US 6780609 B1 20040824 US 2000-543771 20000405
CA 2402410 AA 20011018 CA 2000-2402410 20000621
EP 1268775 A1 20030102 EP 2000-941578 20000621
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL
JP 2004515209 T2 20040527 JP 2001-575181 20000621
NZ 521769 A 20041224 NZ 2000-521769 20000621
US 2003219793 A1 20031127 US 2003-374979 20030228
PRAI US 2000-543771 A 20000405
US 2000-544398 A 20000405
US 1998-71449P P 19980113
US 1998-105511P P 19981023
US 1999-229319 A2 19990113
US 2000-578900 W 20000526
WO 2000-US16951 W 20000621
US 2002-240851 A1 20021004
AB The present invention relates to methods and materials used to isolate and
detect a high bone mass gene and a corresponding wild-type gene, and
mutants thereof. The present invention also relates to the high bone mass
allele, the corresponding wild-type gene, Zmax1, and mutants thereof. The
genes identified in the present invention are implicated in bone
development and in focal adhesion signaling. The invention also provides
nucleic acids, including coding sequences, oligonucleotide primers and
probes, proteins, cloning vectors, expression vectors, transformed hosts,
methods of developing pharmaceutical compns., methods of identifying mols.
involved in bone development, and methods of diagnosing and treating
diseases involved in bone development. In preferred embodiments, the
present invention is directed to methods for treating, diagnosing and
preventing osteoporosis. The invention describes expanded pedigree anal.
and genetic linkage anal. of a high bone mass (***HBM***) gene now
known as an allele of human gene Zmax1. Older individuals with the
HBM allele do not show loss of bone mass compared to normal
individuals, do not have osteoporosis, and do not have any known high bone
mass syndrome. Gene Zmax1 was localized between genetic markers on human
chromosome 11q13.3 and subsequently, BAC clones with the gene were
sequenced. The ***HBM*** allele is inherited as an autosomal dominant
gene and is a G .fwdarw. T mutation at nucleotide 582 in exon 3 which
results in a G171V substitution in the encoded protein. Addnl. genotyping
of 911 individuals established that the ***HBM*** allele is rare and
never found in unaffected individuals. "Silent" SNPs (single nucleotide
polymorphisms) in the gene Zmax1 region were also identified. Gene Zmax1
encodes an LDL-receptor-related protein and the ***HBM*** mutation
occurs in a conserved region of the presumed extracellular domain.
Proteins interacting with the cytoplasmic domain of gene Zmax1 protein in
a yeast two-hybrid assay were identified and include many proteins found
at cell-cell and cell-matrix contact sites. These results suggest a
potential role for gene Zmax1 in focal adhesion signaling and suggest that
regulating gene Zmax1 expression or protein binding may affect bone
processes.
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD

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L10 ANSWER 11 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
reserved on STN DUPLICATE 6
AN 97183437 EMBASE <<LOGINID::20060803>>
DN 1997183437
TI Linkage of a gene causing high bone mass to human chromosome 11 (11q12-
13).
AU ***Johnson M.L.*** ; Gong G.; Kimberling W.; Recker S.M.; Kimmel D.B.;
Recker R.R.
CS Dr. R.R. Recker, Osteoporosis Research Center, Creighton University, 601
North 30th Street 4820, Omaha, NE 68131-5149, United States.
rrecker@creighton.edu
SO American Journal of Human Genetics, (1997) Vol. 60, No. 6, pp. 1326-1332.
.
Refs: 29
ISSN: 0002-9297 CODEN: AJHGAG
CY United States
DT Journal; Article
FS 022 Human Genetics
LA English
SL English
ED Entered STN: 10 Jul 1997
Last Updated on STN: 10 Jul 1997
AB The purpose of this paper is to report the linkage of a genetic locus
(designated ' ***HBM*** ') in the human genome to a phenotype of very
high spinal bone density, using a single extended pedigree. We measured
spinal bone mineral density, spinal Z(BMD), and collected blood from 22
members of this kindred. DNA was genotyped on an Applied Biosystems model
377 (ABI PRISM Linkage Mapping Sets; Perkin Elmer Applied Biosystems), by
use of fluorescence-based marker sets that included 345 markers. Both
two-point and multipoint linkage analyses were performed, by use of
affected/unaffected and quantitative-trait models. Spinal Z(BMD) for
affected individuals (N = 12) of the kindred was 5.54 .+. 1.40; and for
unaffected individuals (N = 16) it was 0.41 .+. 0.81. The trait was
present in affected individuals 18-86 years of age, suggesting that
HBM influences peak bone mass. The only region of linkage was to
a series of markers on chromosome 11 (11q12-13). The highest LOD score
(5.21) obtained in two-point analysis, when a quantitative-trait model was
used, was at D11S987. Multipoint analysis using a quantitative-trait
model confirmed the linkage, with a LOD score of 5.74 near marker D11S987.
HBM demonstrates the utility of spinal Z(BMD) as a quantitative
bone phenotype that can be used for linkage analysis. Osteoporosis
pseudoglioma syndrome also has been mapped to this region of chromosome
11. Identification of the causal gene for both traits will be required
for determination of whether a single gene with different alleles that
determine a wide range of peak bone densities exists in this region.

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